All contributions to be sent to the Editor, 19, St. Andrew's Mansions, Dorset Street, W.1.

British Journal of Anæsthesia

VOL. XIV. No. 4. JULY, 1937.

ANOXIA IN ANÆSTHESIA

By T. A. B. HARRIS, M.B.,
Anæsthetist to Guy's Hospital, etc.

THE Secondary Saturation of McKesson:

"If nitrous oxide anæsthesia is pushed without oxygen or with insufficient oxygen to maintain life a point is reached where the pupil becomes fixed and dilated and respiration slows down and finally ceases. If at this point, or immediately before it, the lungs are inflated with a high percentage of oxygen natural respiration is re-established, the colour returns to normal and it is possible to continue a pure nitrous oxide-oxygen anæsthesia with a very considerable degree of muscular relaxation. This method has some vogue in America, but the general opinion in this country is that it throws considerable strain on the right side of the heart: in any case it should not be attempted by an inexperienced anæsthetist or without a machine capable of inflating the lungs instantly with oxygen under pressure."[1]

"In the majority of patients it is difficult to avoid a trace of cyanosis when pure nitrous oxide-oxygen is being given, and the opinion is gaining ground that in the presence of a free airway this is not detrimental, except in such conditions as toxic goitre, uncompensated heart lesions, etc."

If these two quotations from "Recent Advances in Anæsthesia and Analgesia"[2] are to be taken as an indication of the trend of modern anæsthetic thought in respect to anoxia, then each must bear examination in the light of known and accepted physiological principles.

Nitrous oxide, the weakest of all anæsthetic agents in common clinical use, "belongs to the non-reactive group of gases,"[3] and the members of the group, when inhaled in such a concentration as to exclude oxygen, must, for this reason alone and quite apart from any pharmacological action which they might possess, "act as simple asphyxiant gases."[3]

The first quotation commences with a description of nitrous oxide used as a simple asphyxiant, pushed to such a degree that the resultant anoxia has depressed the medullary centres to the point of respiratory failure, and this is followed by a description of the resuscitation of the patient, before circulatory failure has occurred, by the inflation of the lungs with oxygen under pressure.

Oxygen is almost inert as a physiological stimulant—(it acts only upon the chemico-receptors in the arch of the aorta and the carotid sinus)—but its presence is essential to cell life.

In its simplest form, cell metabolism can be described as consisting of a katabolic phase followed by an anabolic phase.

The katabolic phase is a breaking down process which results when work is performed by the cell, and the end products of this breaking down process is lactic acid. The anabolic phase is a building up process by means of which the cell returns to its *status quo* condition. It is a recovery phase and cell fatigue will result if this anabolic process is incomplete.

The energy necessary to effect this building up or recovery process is provided by the oxidation of the lactic acid formed during the katabolic phase, and the end product of the complete oxidisation of lactic acid is carbon dioxide.

In normal metabolism the amount of available oxygen is always adequate and, consequently, the recovery phase is always complete, cell fatigue does not occur, and the end product of cell activity is carbon dioxide.

Any factor which hinders the complete oxidisation of lactic acid will produce an incomplete recovery phase, cell fatigue will result and the excretory product of cell metabolism will consist of lactic acid in the main, and carbon

dioxide is no longer an accurate measure of the metabolic rate.

Hence, the production of this almost absolute anoxia in the Secondary Saturation of McKesson is nothing more than a rapid production of cell fatigue in such a gross degree that paralysis of the respiratory centre occurs.

Each cell in each organ of the body is subjected to a similar degree of anoxia. Since the cells of the central nervous system show a tendency to resist depressing agents in the reverse order of their evolutionary development, the vaso-motor and cardiac centres escape, solely on account of their greater powers of resistance, and consequently, "the strain thrown on the right heart" mentioned in the above quotation, is merely a measure of the strain thrown on the whole cardio-vascular system as a result of this intense anoxia.

The relaxation of the muscles which results is reminiscent of the flaccid muscles which follow the onset of surgical shock, and in truth, the flaccidity of the muscles, which results when the Secondary Saturation of McKesson is employed, is a measure of the intensity of cell fatigue which results from this intense anoxia. As Haldane has said: "Anoxia not only stops the machine but wrecks the machinery."[4]

The opinion voiced in the second quotation that "in the presence of a free airway a trace of cyanosis is not detrimental" calls for an examination of the significance of cyanosis.

Cyanosis is a measure of the amount of reduced hæmoglobin in circulating blood and this becomes, in normal blood, a measure of the amount of oxyhæmoglobin present.

The minimum amount of reduced hæmoglobin in circulating blood which will produce cyanosis is 5 grammes per cent, which leaves, if the amount of hæmoglobin is normal, 10 grammes of oxidised hæmoglobin per cent.

This is equivalent to an oxygen content of about 13 c.c. per cent, and normal blood, when there is just sufficient reduced hæmoglobin present to produce cyanosis, is, consequently, 67 per cent saturated with oxygen.

It is clear, however, in a polycæthemic patient whose

hæmoglobin is considerably increased, that 5 grammes of reduced hæmoglobin per cent with its resultant cyanosis can readily be present without anoxæmia. In an anæmic patient anoxæmia can conversely be present without cyanosis, for if the hæmoglobin value has fallen below 33 per cent, there is not sufficient hæmoglobin present, even when it is all reduced, to produce cyanosis.

Again, the degree of cyanosis varies with the transparency of the skin, the amount of the blood in the capillaries of the part, the venous pressure and the temperature.

For these reasons, cyanosis, as a measure of the extent to which the storage capacity of the blood is saturated with oxygen, is at best an unreliable guide, but if cyanosis is present, it is more than probable that a diminution in the oxygen content of the blood is present.

While this diminution does not necessarily amount to an actual want of oxygen in the tissues, it does imply a state of affairs in which the oxygen supply from the blood has been reduced to a dangerously low level, and consequently the danger of anoxia is a very real one.

One is justified in saying that except in a polycæthemic patient, if cyanosis is present, a relative degree of anoxæmia is also present, and that there is probably a relative degree of anoxia.

If this conclusion is read into this second quotation, then it is equivalent to stating that except in the presence of toxic goitre and uncompensated heart lesions, etc., in a nitrous oxide anæsthesia a relative degree of anoxia is difficult to avoid, and is not detrimental to the patient.

During the past 20 years an immense amount of work has been done by Warburg, Quastal, and others upon the manner in which tissue cells utilize the oxygen carried to them by the circulating blood.

Oxygen is obtained from the circulating blood, but the blood *per se* never comes into contact with either the source of oxygen supply or the cell which it is to oxygenate. The interchange of oxygen from the blood to the cell is carried out through the intermediation of blood plasma and lymph. Only molecular oxygen, that is, oxygen in simple solution

in blood plasma, is available for cell metabolism, and it can only be used by the cell when combined with cell cytochrome as oxidised cytochrome, or in other similar oxidation systems.

It is suggested that this molecular oxygen is accepted by specific receptors on the cell surface and that these specific oxygen receptors are limited in number, occupy a very small proportion of the area of the cell surface, and vary in nature in keeping with the complexity of the oxidation processes of the living cell.

It is the presence of reduced cytochrome, which only occurs when work has been performed by the cell, that determines the passage of oxygen from blood plasma via the specific receptors to unite with reduced cytochrome to form oxidised cytochrome.

As intracellular oxidation systems are at present inaccessible to therapeutic interference, the only factor over which control can be exercised is the oxygen content of blood plasma, and one can only be sure that blood plasma contains the normal amount of oxygen at its normal tension if arterial blood leaving the lungs is saturated with oxygen to the full extent of its storage capacity.

Should the activity of the specific receptors be reduced, and this can readily occur during an excessive depth of narcosis, the transfer of oxygen from blood plasma to cell cytochrome will be sluggish. In this event an increase in the tension of plasma oxygen may, by increasing the difference of diffusion pressure, in a measure compensate for the inactivity of the oxygen receptors.

Oxygen receptors can be rendered entirely inert in cyanide poisoning, and in this case the oxygen content of blood plasma may be normal or even excessive, but because this oxygen cannot be transferred to the cell in an acceptable form, death of tissue cells from anoxia occurs in the absence of anoxæmia.

Recent workers, notably Hiller[5] and Brinley,[6] have demonstrated that drugs, such as ions, anæsthetics and cyanides, produce their specific action by acting upon the surface of cells and that they do not act in the interior of cells, while Warburg asserts that cyanides and narcotics

inhibit internal respiration by uniting with oxygen receptors and so putting them out of action.[7]

Quastel's work indicates that only oxygen receptors which have to deal with the metabolism of glucose, lactic acid and carbohydrates in general, are occupied by narcotics.[8] He asserts that there is a definite parallelism between the inhibitory power of the glucose oxidation of the brain and the narcotic power of a drug, and the ketosis which follows blood-borne anæsthesia or prolonged narcosis in the treatment of mental diseases would offer some support to this thesis.

The consensus of modern opinion leans towards the view that anæsthetic agents are accepted by specific receptors on the surface of cells, that these receptors constitute a very small proportion of the total cells' surface, that they are limited in number, and probably include those oxygen receptors which have to deal with the metabolism of carbohydrates.

If we accept the work of Warburg and Quastel then the site of this therapeutic absorption is identical with that of oxygen. This hypothesis, however, does not lead to the conclusion that, because some oxygen receptors are occupied by anæsthetic agents, oxygen want must occur, or that oxygen want is identical with anæsthesia.

Oxygen want produces intense cell fatigue, but there is no reason to suppose, even if anæsthesia produced a relative degree of anoxia, that the presence of anæsthesia bears any essential relationship to anoxia. The clinical experience of nearly 100 years has shown only too clearly that anæsthesia is possible with adequate oxygenation of tissue cells.

In anæsthetic overdose a condition of affairs can occur in which the tension of oxygen in blood plasma must be materially increased to render it available for intracellular consumption, and this would indicate either a depression of the medullary centres, producing a stagnant anoxia, and, or, an histotoxic anoxia comparable with the histotoxic anoxia which occurs in cyanide poisoning, produced by the occupation of a more than usual number of cell oxygen receptors.

If the work of Quasel in respect to the site of therapeutic

absorption of anæsthetic agents is accepted, this is the more reason for always having, in anæsthesia, arterial blood leaving the lungs saturated to capacity with oxygen.

In the event of the above view not being acceptable, a lack of intracellular oxygen, whatever be its cause, invariably results in cell fatigue, and, coincident with this, the excretory products of cell metabolism consist of carbon dioxide together with lactic acid and other acid cell metabolites.

Hence, carbon dioxide, which can be looked upon as the universal physiological hormone—for it is the principal regulator of local and general circulatory rate and the degree of lung ventilation—in the presence of an inadequate intracellular oxygen supply can no longer be relied upon as an absolute measure of the rate of metabolic activity.

If the intracellular oxygen supply is inadequate, the three vital reactions, consisting of lung ventilation, the excretion by the kidneys, and the ammonia reaction carried out by the kidneys and liver, are no longer adequate to maintain the pH of arterial blood within physiological limits, and in this event, the physio-chemical reactions, which are in the nature of temporary expedients to remedy a state of emergency, can and do regulate the pH of arterial blood, but at the expense of a gradual and ever increasing degree of cell fatigue, and the ever present danger of an ultimate breakdown of physiological compensation, for the production of acid metabolites which results, throws an ever increasing strain upon the mechanism concerned with maintaining the acid-base mechanism of the body. The compensatory activities of the lung and kidney will be progressively impaired by their own anoxic state, with the result that the reaction of blood and tissue fluids will move steadily towards the acid side and threaten a complete physiological breakdown.

Thus, in the light of known physiological basic principles, it is unsound to suggest that oxygen want should deliberately be called upon to assist anæsthesia, which is in itself a state of emergency.

It is significant that the deliberate use of anoxia in vary-

ing degrees in anæsthesia is a comparatively recent practice and is confined solely to nitrous oxide anæsthesia.

In its lack of deleterious side actions nitrous oxide approaches the ideal anæsthetic agent, but, on the other hand, it is perhaps the weakest anæsthetic agent in common clinical use.

The problem with nitrous oxide anæsthesia is to produce a sufficient depth of anæsthesia and not, as with other more potent agents, that of protecing the patient from the dangers of anæsthetic overdose.

There is a constant striving with this agent to obtain a greater depth of anæsthesia than is to be reasonably expected of it, and this leads, in the first place, to the use of greater degrees of positive pressure than is necessary with more potent agents. The inefficient emptying of the great veins into the right auricle, with the increase of venous pressure which results from the use of such positive pressure, is responsible for the excessive capillary oozing which occurs with nitrous oxide, but in spite of positive pressure, the degree of muscular relaxation obtainable is often inadequate and calls for the assistance of an adjuvant.

It has been seen, in the above quotations, that anoxia is frequently used as the adjuvant of choice in nitrous oxide anæsthesia, but an examination of the physiological principles involved indicates that:—

1. Anoxia cannot be considered an essential corollary of anæsthesia.
2. Anoxia progressively threatens the normal activity of all living cells and can never be considered as offering any assistance to them.
3. Anoxia, employed in anæsthesia, can only be looked upon as an additional burden thrown upon the tissues of the body, already subjected to the unphysiological strain of the anæsthetic agent.

One must therefore conclude that the use of anoxia as an adjuvant in anæsthesia is unsound, while the Secondary Saturation of McKesson is to be heartily condemned.

REFERENCES.

1. McKesson, E. *Proc. Roy. Soc. Med.* (*Anæsth. Sect.*) July 15th, 1926.
2. Macklin, A. H. *Brit. Med. Journ., Aug.* 1st, 1931, p. 212.
3. Henderson and Haggard. "Noxious Gases," 1927, p. 87.
4. Haldane and Priestley, 1934.
5. Hiller, S. *Proc. Soc. Exper. Biol. Med.*, 1927, 24, 427, 938.
6. Brinley, F. J. *Journ. Gen. Physiol.*, 1928, 12, 201; *Proc. Soc. Exper. Biol. Med.*, 1928, 25, 305.
7. Warburg, O. "Uber die Katalytischen Wirkungen der lebendigen Substanz." Springer. Berlin.
8. Quastel, J. H., and Wheatley. *Proc. Roy. Soc. Med.*, 1932, cxii, 60.
   Quastel, J. H., and Davis. *Journ. Biochem.*, 1932, xxvi, 1672.
   Quastel, J. H. *Lancet*, March 4th, 1933.

TECHNIQUE OF CARBON DIOXIDE ABSORPTION METHODS

By E. G. Van Hoogstraten, M.B., Ch.B.,

Clinical Teacher in Anæsthetics, University of Cape Town, South Africa.

CAREFUL perusal of the numerous articles and text-books now being published on "Modern Anæsthesia" leaves one amazed at the comparatively slight prominence given to carbon-dioxide absorption methods in nitrous-oxide administration. Until the advent of cyclopropane, the high cost of which compelled the adoption of absorption technique, the method appears to have received but little of the support it merits.

It is to be presumed that with the cost of nitrous oxide as low as it is in England, it was deemed hardly worth while departing from the hitherto accepted procedures. For those, however, whose lot it is to practise in the Colonies, where cost, availability, storage, and transport are serious problems, my experience might be of some interest and help.

In South Africa nitrous oxide costs 18*s*. per 100 gallons, constituting quite a big item in the annual budget, and therefore the greatest drawback to its routine adoption for general hospital use. In private, apart from the expense and the inadequacy of fees, the inconvenience of having to cart, from case to case, sufficient gas for an operation, however long, in addition to his apparatus, did not add to the anæsthetist's enthusiasm for "safer and pleasanter anæsthesia".

A recent visit to America, made chiefly to get first-hand experience and information on cyclopropane, went far towards solving one's problems. There I received my initial

introduction to the absorption technique. First by Waters, of Madison, in his pioneer work on cyclopropane, and later at the Mayo Clinic, where the principle was applied to nitrous-oxide administration. The tremendous economical saving was at once apparent, but even more striking were the results obtained by the nurses, who administer the general anæsthetics. Even in robust, abdominal cases, they seemed to have no difficulty in getting excellent relaxation with N_2O, O_2, and *truly minimal* ether, rarely exceeding one ounce of the latter.

The machine they used was a "Heidbrink Kinetometer," specially built to the specifications of Dr. John Lundy, senior anæsthetist to the Clinic. Truly a remarkable machine, fulfilling every want, and one the manufacturers can be proud of. Sturdily and compactly built, it incorporates all the features of a "perfect volumetric multiple-gas" apparatus. It is practically fool-proof, will stand up to any amount of hard work, and has no intricate parts to "go wrong".

The flow-meters are of the dry, sight-feed type, calibrated in "gallons-per-hour", and litres-per-minute", for nitrous oxide, cyclopropane, carbon dioxide, and oxygen. They are extremely simple in design, consisting of a tapered tube (with float), through which the gas passes in volume as regulated by a release valve operated by a thumb screw. As the gas passes through the tube it causes the float to rise, and accurately register the *volume* being delivered to the patient. In addition, a self-closing emergency-flow, operated by a push lever, is fitted for each gas, excepting the CO_2 and cyclopropane. These provide for a rapid filling, or change of the mixture in the bag, and, in the case of the oxygen, permit of rapid inflation of the lungs, at pressure, in emergency.

An exceedingly ingenious and practical "circle absorber" is fitted to the machine. By simple lever adjustment, all, of any part of the exhaled gases, may be directed either through the soda lime, or around it. To provide for rapid CO_2 absorption and quick results, the absorber operates to pass the breathed gases through the soda lime, both on exhalation and on inhalation.

A lever operates to permit emptying of the bag without having to remove the mask from the patient's face, and also provides for the instant administration of pure oxygen under sufficient pressure for lung inflation. All fresh gases are delivered directly into the inspiratory tubing, thus ensuring immediate response by the patient to any change in the anæsthetic dosage. Flutter valves operate freely and with practically no resistance to breathing.

The container is divided into two, so that should one lot of soda lime become saturated during the operation, the anæsthetist can merely switch on to his reserve chamber, without having to detach and refill. The chamber is of 12 to 15 hours' capacity, and is quickly removable for refilling when necessary. An excellently designed ether bottle completes the whole ensemble.

Another very fine machine is the Foregger Metric, as used by Dr. R. Waters, of cyclopropane fame. He prefers to use a simple "to-and-fro" filter, close to his mask, thus avoiding the inertia of the gases in the tubes leading from the mask to the circle filter. The flow-metres act on the principle of *displacement* of water by differential pressure calibrated in volume per time unit. The reading of the meniscus is facilitated by floaters.

For accuracy it has probably no superior, but on account of the large volume of water in glass containers, it is more adapted to hospital use, than to stand up to the hard knocks of continual transport, etc. In case of breakage, especially in the Colonies, replacement might prove a difficult and tardy matter, as the gases are individually calibrated and the scales are only made upon construction of each individual machine.

This having to be my first consideration, I invested in a Heidbrink machine, and have never regretted the outlay. Within a year it had paid for itself in the saving of gas alone; on an average it was possible to get from 12 to 15 anæsthetics of however long duration out of a 100-gallon cylinder of N_2O, as opposed to the 80–100 gallons per hour necessary in non-absorption methods.

A small, easily portable, 50-gallon cylinder provides sufficient gas for several anæsthetics. One can even imagine

a sparklet size cylinder being sufficient per operation, and ultimately replacing the cumbersome "bombs" the anæsthetist still has to carry about.

Apart from the saving and the convenience, the resulting anæsthesia surpassed all expectations. No longer was it necessary to try to "educate" an exacting surgeon to be satisfied with a merely "operable" abdomen, or else put up with sarcastic remarks *re* the abdomen being as "tight as fiddle-strings"!

The admission of rarely more than on ounce of ether produced ample relaxation; in favourable subjects almost equivalent to that of a spinal anæsthetic. Breathing was quiet and effortless, conserving the patient's energy, body heat, and moisture, and a great boon to surgeons in abdominal work. Evidently the removal of carbon dioxide lessens the tonicity and the responsivity of muscles to stimuli, just as CO_2 accumulation during sleep increases it, and is said to explain such phenomena as colics, onsets of appendicitis, labour starting, etc., usually occurring at night.

In fact the question arises whether a great deal of the relaxation and quiet breathing obtained with cyclopropane is not largely due to the CO_2 absorption methods used in its administration. Certain it is that the results obtained in abdominal work, using "absorption" and "non-absorption" techniques respectively, cannot be compared. In the latter, "minimal" ether is usually a very elastic term, unless previously augmented with intense premedication as avertin or pentothal sodium, or else by local or spinal injections. With the absorption technique, this is very seldom necessary, excepting in the most robust types of cases. It is indeed rare to have to use more than one ounce of ether.

It is almost possible to predict that, once accustomed to absorption technique, no anæsthetist will willingly revert to non-absorption methods, especially for abdominal work. An exception perhaps is when dealing with very young children. Their metabolic rate is comparatively higher than in adults, and is more difficult safely to control with omnopon-scopolamine, etc. Their low tidal volume of respiration renders it difficult for them to overcome the inertia

of the gases in the tubes between the circle filter and the mask, which therefore circulate inadequately, and build up a higher percentage of CO_2, to which children are very susceptible. Here non-absorption methods seem to act best, or else a simple "to-and-fro" filter next to the mask should be used.

Experience shows that the best results are obtained by using Wilson's 4–8 mesh soda lime, the grade and mesh being of importance.

Absorption technique simplifies the administration of anæsthetics tremendously. Teaching experience shows that even a student can become reasonably adept and "safe" within a week, using absorption gas-oxygen-ether sequence, whereas it takes months before he can really be entrusted with chloroform-ether, or before he can be relied upon to produce an operable abdomen with gas-oxygen, or even gas-oxygen-ether, by non-absorption methods.

Unlike the McKesson, Volumetric machines do not work on the principle of percentages, nor are they capable of delivering the gas at adjustable positive pressures. Hence the necessity of some ether, however minimal, to get a smooth prolonged anæsthetic, must frankly be admitted. However, this is not a serious drawback. Indeed, it remains a moot point whether it is not preferable to attain one's ends by frankly allowing minimal ether, than by temporarily turning the patient into a C_3 risk with primary and secondary saturation. Increasing experience has brought to the writer a greater tolerance for ether; rather "balanced" anæsthesia, than going to extremes with any one particular agent.

The great factor is to get a smooth, rapid, safe induction, without preliminary saturation of the patient with toxic lipoid-soluble substances. Once this is attained there seems to be very little difference afterwards, as far as vomiting, shock, chest complications, etc., are concerned, between gas-oxygen, and gas-oxygen-ether sequence. The patient may remain asleep a bit longer, but this is all to the good.

Absorption methods bring the advantages of gas-oxygen-ether combination within the pocket of every patient, and within the scope of every anæsthetist. In expert hands, for

straight gas-oxygen work, the McKesson has no equal, but for the less experienced, or once the necessity for ether, or for a mixture of gases, is admitted, volumetric machines will probably prove preferable.

Percentage scales are good enough as a guide, especially if only N_2O and O_2 is being used, but they cannot furnish correct quantitative readings. With volumetric machines one gets anæthesia with known amounts. It is anæsthesia with definite knowledge of every detail of the performance. The necessity of a reliable way to adjust the proportion of gas mixtures is brought home in particular by the advent of ethylene and cyclopropane, which gases need oxygen in far larger proportions than does nitrous oxide. The oxygen percentage again readjusts itself when a mixture of nitrous oxide, ethylene, or cyclopropane is used.

Knowledge of the amounts of the anæsthetic gases is of importance, if respiratory volume is granted the position of basic factor in inhalation anæsthesia, and if consideration is given to rate of absorption, saturated amount, volability, and similar conditions in relation to the blood.

Carbon-dioxide absorption method especially necessitate a fine and definite oxygen adjustment to feed at a metabolic rate of minimum 100, and maximum 500 c.c. per minute. The principle involved in this method is the respiratory quotient during anæsthesia, which is worthy of study. Without the possibility to replace O_2 in measured amounts for all CO_2 given off, the method would be an economic and practical improvement, with incomplete scientific support.

To summarise, one feels that CO_2 absorption methods have definitely come to stay. In abdominal work they are a boon to anæsthetist and surgeon alike, rendering it easy to attain good relaxation, quiet, effortless breathing, and help in conserving the patient's energy, body heat, and moisture.

Especially in the Colonies it is a tremendous economical saving and solves the problem of portability, storage, transport, etc.

The volumetric machines in use enable the anæsthetist to observe, adjust, and measure the flow of all gases used, and gain a true picture of the anæsthetic performance; of

special value when supplementing N_2O with cyclopropane, etc.

Practical points are: (1) A complete free airway. (2) Apparatus, mask, etc., completely without leak. (3) The use of the right type of soda lime. (4) Suitable apparatus of proper mechanical construction to avoid resistance to breathing while permitting sufficient contrast to atmosphere with alkali completely to remove carbon dioxide from the inspired gases.

As "ideal" machines may be quoted the Heidbrink "Kinetometer" and the Foregger "Metric".

In conclusion, my sincerest thanks and acknowledgment are due to Doctors Heidbrink, McKesson, Foregger, and Waters, whose kindness and help have proved of such inestimable value in solving one's problems. They are true pioneers, only too willing to pass on their knowledge. Their inventive genius in producing and perfecting the machines of to-day have gone far towards rendering possible and putting modern gaseous anæsthesia on the plane it now occupies.

CASUAL COMMENTS

IT was recently our good fortune to listen to the conversation of an elderly anæsthetist, of long experience in his art and of sound judgment. Being of an eager turn of mind, he had always kept abreast of knowledge in his own subject and had adopted, when he had not initiated, new methods as they evolved from practice. It was not surprising that a man of this kind had arrived at definite opinions on a variety of those questions which frequently trouble the minds of less experienced men. These he treated us to in a number of aphorisms, some of which we reproduce later on, as well as in the general run of his remarks. Although, as we have indicated, expert in the use of gaseous anæsthetics and in the handling of the chief machines by means of which nitrous oxide and cyclopropane and ethylene are instilled into their fortunate recipients, this man still had much use for ether and even for chloroform. The latter he regarded as still the most generally useful anæsthetic for obstetric practice, for which purpose he maintained that it should be used only from some apparatus which is foolproof as regards dosage. Capsules he derided. "What's to stop a nurse overdosing with capsules?"

Some of this elderly gentleman's most telling experiences were related in connexion with the accidents of anæsthetic practice. Accidental burns of various kinds and causes had much impressed him in the course of his many years of practice. Those directly due to the anæsthetic have, of course, disappeared with the disappearance of the "rag and bottle" administration of chloroform. Burns due to modern heated tables and careless protection of the unconscious patient, as well as those from hot bottles are still to be met. In this connexion our old friend observed: "Never accept a nurse's statement that the hot-water bottle in a bed is cold".

Another of the "nevers" which he had formulated for himself in the course of time was this: "Never have a chest X-rayed for the missing tooth until you have examined thoroughly the turn-up of your own and the dentist's trousers!" And another, which many men would laugh at, but which is really sensible, was this: "Never treat lightly the statement of the patient who is sure she will die under the anæsthetic!" Perhaps the most valuable of all his "nevers" was "Never be late for an operation."

Like all anæsthetists who were, so to speak, brought up on ether, he had been at great pains to find some means of saving his patients from the unpleasant taste and odour which used to hang about those who had endured a long inhalation. He stated that essence of peppermint, or bitter orange, would both mask the smell of ether, and that for a mouth-wash after operation either brown vinegar and water, or Schweppe's ginger ale, were excellent.

Talking of the minor inconveniences that were sometimes associated with the machinery of modern anæsthetic apparatus, the narrator observed that condensation of moisture is often a nuisance on the ether bottle of gas and oxygen machines which have these attachments. A mixture of equal parts of glycerine and water smeared over the outside of the bottle effectually prevents this particular annoyance.

The Australian Society of Anæsthetists, actuated by a laudable desire to solve the problem of ether convulsions, issued a questionnaire which was replied to by fourteen anæsthetists. Their answers related to some 81,700 administrations. They disclosed the fact that there were convulsions during anæsthesia when ether was not employed besides those rightly described as "ether convulsions". The Society accordingly considers the two classes of convulsion "ether convulsions" and "convulsions during anæsthesia" separately. This is wise, for it does not appear that the "convulsions during anæsthesia" are exactly similar to those which occur in

ether narcosis. For instance no experienced anæsthetist could compare anoxæmic convulsions during "gas" anæsthesia with the ether phenomenon. One of the non-ether cases recorded by the Society is especially interesting because the patient's temperature on return to bed after removal of a hastily inflamed appendix was 107.4°F. This patient had evipan and nitrous oxide and oxygen. "Tremor of limbs was noticed two or three times during operation." The temperature was intermittent, after the hyperpyrexia had been reduced by rubbing the body with ice, but not above what could be explained by the abdominal condition. Recovery was complete. The diagnosis was held to rest between heat-stroke, malaria, cerebral embolus, and a pontine hæmorrhage.

In another instance of convulsions during ethylene anæsthesia in a patient undergoing operation for compound fracture of tibia and head injuries, the surgeon suspected a cerebral injury. He trephined but found nothing to account for the convulsions, which were attributed to the anæsthetic. Some of the other instances of non-ether convulsions occurred after injection of local anæsthetics. These were either cocaine or allied substances, all of which are well recognised as possible causes of convulsion in susceptible individuals.

There were only four instances of ether convulsions described in the answers to the Society's questionnaire. In three of these the anæsthetic was given endotracheally. The four patients all recovered, and there is nothing in the account of their convulsive attacks which sheds any new light on the problem of what causes this phenomenon. Each of these instances could be almost exactly matched, in fact, by cases which have been recorded in our own pages or in other medical journals in this country.

CONTRIBUTIONS TO THE STUDY OF ETHER CONVULSIONS

RECORDS of instances of ether convulsion continue to reach us, and in the following pages we reproduce our most recently received contributions to this perennial puzzle. It appears that this complication is more often met in England than elsewhere; but there is no obvious reason for this any more than there is for the other peculiar features of the phenomenon.

Ether Convulsions Occurring Twice in the same Patient.

By Z. Mennell, M.D.,

Senior Anæsthetist, St. Thomas's Hospital.

1st Operation:—Cholecystotomy.

Patient, a woman aged 42 years. Fat, extremely ill, high fever, rapid pulse—very toxic.

Anæsthetic: Induction N_2O-ether sequence after atropine gr. 1/75. Followed by ether on mask and oxygen blown through ether. Phillips air-way. Induction easy: operation difficult, the surgeon requiring complete relaxation, which was easily obtained. As the abdomen was being closed at the end of about 50 minutes, the typical twitching at the corner of the mouth and eyelids occurred. This rapidly spread to the limbs and abdominal muscles. Anæsthetic stopped and morphia gr. $\frac{1}{3}$ given. The back rest was lowered and Trendelenburg position substituted. Twitching soon stopped and patient made an uninterrupted, though from the sepsis an inevitably rather stormy recovery.

2nd Operation: Six weeks later.

Cholecystectomy: extremely difficult. Same anæsthetic, the same result; but as the operation had not been com-

pleted when the first sign of twitching occurred an intratracheal catheter was passed immediately and the anæsthesia continued with N_2O and O_2 to which was added $CHCL_3$ to retain complete relaxation. Morphia gr. $\frac{1}{4}$ given on table. The gall bladder rest had to remain and slight Trendelenburg position used. The operation had to be stopped for some five or six minutes and then continued for over half an hour without trouble. Convalescence uneventful.

The points about this case are:—

1. Severe sepsis and toxæmia at the time of the first operation.
2. Condition at the time of the second operation after six weeks' drainage of gall bladder extremely good.
3. The weather was neither hot nor cold.
4. The temperature of the theatre not abnormal. There was no undue sweating.
5. The patient was fat, the operation difficult, a very deep anæsthesia essential and a large quantity of oxygen was used.
6. The condition was recognised at once.
7. The air-way was clear with a bright pink colour and shallow respiration when the convulsive movements commenced.
8. The vocal cords were widely dilated when the large catheter was passed.
9. CO_2 was given at the first operation with no result. Not given at the second.
10. There was negligible vomiting after the first operation —none after the second.
11. On each occasion muscular stiffness was complained of; more than that usually associated with the use of the gall bladder bridge.
12. The question of a spinal anæsthetic for the second operation was considered carefully and rejected.

Should a third abdominal operation be necessary, as seems possible, what anæsthetic should be used?

I am inclined to think an intravenous barbiturate and intratracheal gas-oxygen-ether sequence.

Two Instances of Ether Convulsion.

By Ashley Daly, D.A.,
Senior Anæsthetist, London Hospital.

The subject of ether convulsions has been discussed more than once at the Section of Anæsthetics, Royal Society of Medicine, and a good deal has been written about it in the medical journals, but in spite of this we have no knowledge as to the exact cause of the condition, or the treatment we should adopt when it arises, and so I need make no apologies for bringing forward the notes of two cases of this condition which occurred recently at the London Hospital.

For the notes of Case 1 I am indebted to my colleague, Dr. N. A. Gillespie, who also kindly called me from a neighbouring theatre to see the patient when the convulsions had developed. She was a married woman, 36 years of age, who had a laparotomy for obstructive jaundice on October 27th, 1936. In the previous January, that is nine months before, she had had Caesarean section performed under general anæsthesia at another hospital, chloroform being the agent employed, and there is a story of difficulty with the anæesthetic, rigidity and cyanosis persisting throughout. Following this there was a period of normal health for a few months, after which gradually increasing jaundice developed and she was admitted to the London Hospital.

At the operation in October the anæsthetic was ethyl chloride followed by ether on an open mask, atropine gr. 1/100 being the only premedication. She was not intubated; an ordinary Phillips' air-way was used. The patient had been having 5 c.c. of calcium gluconate twice a day intravenously for some weeks. The operative procedure was laparotomy with removal of a piece of liver for examination.

The induction and early stages of the anæsthesia were uneventful, but with the onset of deep anæsthesia, cardiac irregularity and later tachycardia developed. This was followed by typical ether convulsions just as the peritoneal

closure was completed. CO_2 and oxygen were given at once and the head and shoulders raised and the feet lowered, at first with no success, but after a few minutes the convulsions suddenly ceased just as a dose of evipan was about to be put into a vein. The convulsions did not recur. There was no post-anæsthetic vomiting, but slight bronchitis developed.

After-history. The patient gradually went downhill. After three weeks ascites developed and she died one month after the operation. Post-mortem examination revealed general fibro-sero-purulent peritonitis and acute yellow atrophy of the liver.

Points in this case to which attention may be drawn are: (1) The weather was not hot, but the patient had been running temperatures round about 104°. (2) She had had a previous abdominal operation, chloroform being used. (3) She was deeply anæsthetised at the time convulsions started. (4) The convulsions ceased spontaneously after the head had been raised for a few minutes and just before evipan was to be given. (5) She had been having calcium gluconate before operation.

Case 2. For the notes of this case I am indebted to Mr. B. B. Hickey, a House Officer at London Hospital. The patient was a male child aged $3\frac{1}{2}$ years, admitted to hospital at 12.15 a.m. on March 3rd, 1937, with a three days' history of abdominal pain and vomiting. Eight hours before admission symptoms became worse, the breathing being rapid and embarrassed. On examination the child looked very ill, face flushed, temperature 101.4, pulse 140, respirations 32.

Diagnosis. Acute appendix.

Operation. 1 a.m., atropine gr. 1/150 at 12.40.

Anæsthetic. Gas-oxygen-ether from Boyle's apparatus. Induction easy, rapid and no cyanosis.

Operative findings. Acute perforated appendix. Pelvic peritonitis with abscess formation. Perforation of loop of small intestine which formed part of wall of abscess.

Procedure. Appendix removed, perforation sutured. Two drains inserted. Operation lasted 25 minutes.

There was no trouble with the anæsthetic for 20 minutes when, as the peritoneum was being closed, the face twitched and the abdominal muscles became rigid. This lasted a few seconds and the child became cyanosed. The ether was at once shut off and the boy was given CO_2 added to oxygen. Breathing and colour quickly became normal and the operation was completed without difficulty.

On removing the mask the child again became cyanosed but this was at once corrected with pure oxygen. As the patient was about to be removed from the theatre cyanosis reappeared. The pupils dilated widely, there was spasm of the jaw muscles and general clonic convulsions began. Oxygen with about 12 to 15 per cent CO_2 was given, and after a few breaths of this mixture the convulsions ceased, only to recur again on removing the mask, but again to be abolished by inhalation of the mixture. A little later, however, a convulsion occurred whilst the mixture was being inhaled. These convulsions consisted of about 10 clonic spasms of the flexors of the limbs and digits, generalised facial twitchings with clonic movements of the neck and jaw muscles, synchronous with the flexor movements of the limbs. The convulsive movements were accompanied by short gasping inspirations. This was succeeded by a varying number of normal inspirations, after which the train of events recommenced. Cyanosis was avoided if the CO_2 oxygen mixture was given during the convulsions.

The individual movements of the convulsions were predominantly clonic contractions of the flexors; there was no pronation of the forearms and no rotation of the hips; the fingers became adducted but not to a greater extent than is normal in flexor movements; the thumb was not opposed, it was flexed with the rest of the fingers. It was agreed that the movements and postures of the limbs did not resemble those seen in tetany.

The CO_2 oxygen mixture was continued during the journey back to the ward, but on removing the mask to lift the patient into bed cyanosis at once reappeared. Morphia gr. 1/12 was given. The convulsions continued unchanged for about 40 minutes when the pulse, which, though rapid, had continued steady, became weak. Cyanosis persisted in

spite of oxygen, the convulsions became weaker, and the child died at 2.40 a.m.

The methods of treatment tried in this case were: (1) Varying mixtures of CO_2 and oxygen. It was found that a mixture containing 12 to 15 per cent CO_2 in oxygen had most effect. (2) Changing position of patient. He was definitely worse when foot of bed was raised. Very slight improvement was gained with high Fowler position. (3) Injection of morphia. Examination of the ether employed showed no impurity.

Here, then, we have two patients of different ages and sex showing the same symptoms, though of different severity, at the end of an abdominal operation under deep ether anæsthesia.

In the *British Journal of Anæthesia* for July 1936 there are two papers dealing with the subject. In one paper Hoseason puts forward the idea that the condition is due to calcium deficiency, and he quotes a case of ether convulsions which responded dramatically to an intravenous injection of calcium, but in the first case which I report, the patient had been having intravenous calcium twice a day for weeks and can hardly have been suffering from calcium deficiency.

In the other article Vaughan Hudson reports two cases and quotes freely a paper published in the *Lancet* by Woolmer and Taylor. Hudson's first case was a typical one, a child with a gangrenous appendix who developed fatal convulsions towards the end of the operation. It was noticed in this case that the child's skin was extremely hot and the axillary temperature was found to be 106°.

Intravenous calcium was given without effect.

The second case was that of a child of two months old who was operated on for inguinal hernia under local anæsthesia, novocain .75 per cent being used. The convulsions occurred during a fairly severe surgical stimulus, viz. the separation of the cord from the wall of the sac. In this case the operation was rapidly completed and chloroform was given. This caused both respiration and convulsions to cease, but gentle pressure on the chest started the respirations and the convulsions did not recur, the child making a good recovery.

Hudson puts forward the view that the cause of the convulsions is surgical trauma in the presence of too light a degree of anæsthesia, and he suggests that with the onset of convulsions all forceps and instruments likely to provoke stimuli should be removed, and that the anæsthetist should deepen the anæsthesia by the most rapid means at his command, preferably with evipan, or failing this, with chloroform.

As regards the first suggestion it is my experience and the experience of a good many people I have spoken to that where convulsions come on the anæsthesia has always been very deep, in fact too deep, after showing that jerky breathing which one associates with over-deep ether anæsthesia. It would seem to me to be a very dangerous procedure to give the patient chloroform; fatalities *have* been recorded from this method of treatment. In a rigid, blue patient, taking occasional gasping breaths, it must be very difficult to regulate the intake of chloroform.

Evipan would appear safer. as it would be easier to regulate the dose, though the difficulty of finding a way into a vein in these patients must be considerable.

I should prefer to try methods which aim at rapid elimination of the anæsthetic, which relieve cerebral congestion and lower temperature.

In provoking discussion I would suggest that there are many factors which cause this condition, some of which are (1) sepsis, (2) pyrexia, (3) hot weather or very hot theatre, (4) deep anæsthesia, (5) cerebral congestion, (6) I have an idea that atropine may have something to do with it, but I shall be told that atropine has been given to hundreds of thousands of cases without causing convulsions. I would like, however, to ask these two questions: (i) Has anyone had a case of convulsions in which atropine had not been given? (ii) Has anyone had a case of convulsions when morphia had been added to the atropine?

I think it was about 1908 or 1909 that atropine began to be used, and certainly before that no case of convulsions had been reported.

(7) Surgical trauma. Most cases seem to occur when the peritoneum is clipped with forceps preparatory to being

sewn up. As regards treatment I suggest: (i) Withdraw anæsthetic and hasten elimination with CO_2 and oxygen; remove forceps. (ii) Raise head and lower feet to relieve cerebral congestion (steeply). (iii) Apply cold pack to head and neck to lower temperature. (iv) If these measures fail give intravenous evipan.

Ether Convulsions Controlled by Evipan.

By H. Grantham Dodd, M.B., B.S., D.A.

T.C.D., male, aged 37 years, a steel-worker of average muscular development, but with a large, prominent muscular, and deep abdomen, was admitted to the Llanelly General Hospital during the evening of May 25th, 1937.

He had had an attack of abdominal pain about a fortnight previously, without vomiting; this had eased off, and he had been at work since. At five o'clock on the afternoon prior to admission the pain had recurred, commencing at the xiphisternum and going down to the umbilicus, and had gradually got worse; he went to work as usual on nightshift. On the morning of the 25th he took a fruit laxative, and his bowels were opened. The pain got much worse at 3 p.m., and he vomited slightly thrice.

He was admitted to hospital with a temperature of 100.2, pulse-rate 96, respiration 26, and a furred tongue. There was general rigidity and tenderness of the abdomen: there appeared to be definite loss of liver dullness, but owing to the configuration of the abdomen this sign was not considered to be of much value: there was gurgling on auscultation to the left of the middle line over the gastric area.

He was taken to the operating theatre at 10 p.m. for operation by Mr. D. McLennan, F.R.C.S.

The Anæsthetic. A dental prop was inserted between the lower teeth and the edentulous upper jaw. Induction was by C_1E_4, C_1E_{32}. Ether sequence with an Ogston's mask. There was no difficulty during induction; no obstruction nor cyanosis; deep surgical anæsthesia was obtained, with the pupils definitely commencing to dilate. At this stage a No. 8 Magill's rubber endotracheal tube was

passed with ease orally, and adjusted. A Boyle's apparatus was turned on with three-quarters blow-over of the small ether bottle, using a face mask and Clausen's harness. The operation was commenced at once with a right paramedian incision through the skin and muscles. By this time the patient was certainly not in a very deep stage of anæsthesia.

Almost immediately muscular twitching commenced about the eyes and forehead, spread to the lower face, then became general. The ether was turned off first, then the N_2O, and O_2 and CO_2 continued alone. The muscular spasms decreased, but did not entirely cease: cautious re-administration of ether caused a recurrence, and the ether was again turned off.

The problem now was how to proceed: the operation was one of necessity, and it was quite evident that N_2O and O_2 alone would be insufficient. An attempt was made rapidly to push the patient with the help of CO_2 to deep anæsthesia, in the hope that when this was achieved the neuro-muscular mechanism would be completely depressed, resulting in the cessation of the convulsions. This object proved, however, to be impossible of accomplishment; the convulsions became so violent that, not only had the surgeon to lean with his weight on the lower abdomen and thighs to control the patient on the table, but respiratory efforts became irregular and ineffective. Ether was discontinued and a solution of evipan sodium rapidly prepared. A vein in the dorsum of the foot was entered with some difficulty, and a dose of 3.5 c.c. administered. No attempt was made to time the rate of injection, but it was given rather quicker than the regulation 15 seconds per c.c. The convulsions promptly ceased, and the anæsthetic was continued, using N_2O and O_2, ether, and CO_2.

The operation was now continued, and the resulting appendicectomy proved to be one of the longest and most difficult at which I have officiated. On opening the peritoneum an enormous omentum presented, infiltrated with a dense layer of solid fat, and occupying a large part of the abdominal cavity. The lower end of the omentum was enclosing the appendix, which had two perforations near

the base with a thin bridge of tissue between them. The adjacent portion of the cæcum was inflamed and rotten; it was entirely immobile, and with the appendix was rotated mesially. The under surface of the omentum was more or less bathed in pus, and there was much pus in the pelvis, not markedly offensive. The local conditions, together with the barrel-shaped abdomen and a thick layer of superficial fat, made the work of removing the appendix and the attempt to bury the stump one of extreme difficulty.

In order to assist the surgeon as much as possible no attempt was made to spare the ether, an abnormal amount of which was used. About 6 ozs. was used from the small bottle, and an unmeasured amount was added from time to time from the large bottle. The rectus abdominis muscle was noticed to be quite flaccid during the operation.

Towards the close the patient's face was noticed to be burning hot to the touch; the temperature taken in the axilla before leaving the theatre was 104.1. A dose of 3.5 c.c. of coramine was given intramuscularly.

Next morning the patient felt and looked fit, and his temperature was normal. He never reacted, however, got more and more toxic, there was severe sloughing of the operation wound, he developed hiccough and diarrhœa, complete anuria supervened on the ninth day, and he died on June 4th.

In view of the varied suggestions which have been put forward from time to time by numerous observers to account for this strange condition, the present case provides several points of interest.

(1) Atropine, idiosyncrasy towards, or relatively large dose of. Preliminary medication consisted of morphia gr. $\frac{1}{6}$, atropine gr. 1/60. This is the dose I am in the habit of prescribing in the case of robust men. Cocaine was not employed prior to intubation. Curiously enough the condition has also been ascribed to the omission to use atropine. Convulsions are not a feature of atropine poisoning, although they have been recorded in the case of infants and young children.

(2) Impurities in the ether. Ether had been in use as an anæsthetic for 80 years before "deep ether convulsions"

were recognised and described. It can hardly be doubted that the ether in use at the present day is of a much higher grade of purity than that available during the earlier period. At the termination of the operation the ether remaining in the bottles of the Boyle's apparatus was deliberately left for use on the next day, fresh ether being added to the small bottle immediately before induction in sufficient quantity to bring the total up to four ounces. The first case on the list was one for perineal repair, and during the administration the artificial air-way was from time to time so adjusted as to allow of partial obstruction from falling back of the base of the tongue with a slight but definite degree of cyanosis: no untoward phenomena occurred, nor was any expected. The ether used was the well-known brand supplied by the British Drug Houses.

(3) Presence of anoxæmia. The methods employed and the ease of administration in the present case render this most unlikely.

(4) The use of CO_2. No CO_2 had been used prior to the onset of convulsions. An accumulation of the patient's own CO_2 does not occur when an endotracheal tube is employed.

(5) Deep and prolonged anæsthesia. Convulsions started at the commencement of the operation. The patient was not unduly deeply anæsthetised, though deep anæsthesia had been present for a few moments before intubation. In many of the reported cases the operation has reached an advanced stage, and after the convulsions have been controlled, it has been hurriedly completed without any further anæsthetic, or with N_2O and O_2 alone. In the present instance the operation was continued at leisure, and relatively large amounts of ether were used unsparingly.

(6) Hurry to get a seriously ill patient quickly out of the theatre, with consequent trauma to the peritoneum and to the vessels of the mesentery. The peritoneum had not been opened.

(7) Septic conditions with toxæmia, especially intra-abdominal. This patient fulfilled these conditions completely.

(8) A very high theatre temperature. This theatre is heated by steam pipes with very inadequate control. The

atmosphere is frequently oppressive, sometimes to an almost unbearable extent. On this night the temperature of the theatre, with two windows open, was 85°F.

(9) Deficiency of the blood calcium. On the day following operation a sample of venous blood was collected and despatched to Professor E. C. Dodds, of the Courtauld Institute of Biochemistry, the Middlesex Hospital, who very kindly undertook an investigation of the blood calcium. His report is as follows: Calcium, 9.8 mgs. per 100 c.c.; phosphates, 3.7 mgs. per 100 c.c.; and he adds that this figure is within normal limits, these being from 9 to 11 mgs. per 100 c.c.

(10) It has been stated that patients who have recovered from ether convulsions pass urine which is strongly alkaline in reaction for several days afterwards. This patient's urine on the second morning after operation was strongly acid.

Calcium in Ether Convulsions.

By J. U. Human, M.R.C.S., L.R.C.P.,
Honorary Anæsthetist, Queen Mary's Hospital for the East End, etc.

The reason for reporting this single case of late ether convulsions is that several years might elapse before I encounter another case, and that other anæsthetists might be encouraged to try this method and report their results. A 10 c.c. ampoule of calcium takes up very little space in one's bag and to the best of my knowledge it will keep indefinitely.

A well-built, muscular youth, aged 19 years, was admitted to hospital for simple malunited fractures of the radius, ulna, and humerus on the right side, and the operation was for plating of the bones.

A 1/100-gr. atropine sulphate was injected half an hour before and induction was by means of a nitrous oxide, oxygen, and ether sequence. A Boyle's type dry bobbin flow-meter machine was used throughout.

The patient required rather more than the average

amount of anæsthetic before relaxation was obtained, and relaxation was necessary for aligning the over-riding fragments.

At the end of 1¼ hours respiration became jerky and shallow and some duskiness appeared. No twitching of the eyelids was noticed at this time, but on removing the mask twitching of the lips was noticed.

The mask was strapped on again, and pure oxygen given. The colour became bright pink in a few seconds, but as the convulsions became increasingly severe 10 c.c. of a 10 per cent solution of calcium gluconate (Sandoz) was injected intravenously in the left arm, and within one minute the convulsions suddenly ceased. They had been so violent that it was with difficulty that the intravenous injection was made at all.

Nitrous oxide and oxygen only was used for the rest of the operation, which took another three-quarters of an hour to complete. The patient had no further convulsions and made a good recovery.

It is difficult to estimate time when this sort of thing happens, but I am sure that from the onset of convulsions until the finish did not take more than five minutes.

Elimination of ether could not account for the cessation of convulsions as the patient was still well anæsthetised when convulsions stopped.

There was no sepsis in this patient, and adequate rebreathing was employed throughout so that there was no loss of CO_2 through hyperventilation.

In this patient, therefore, a deficiency of calcium seems to be the only pathology that will explain the events satisfactorily.

It is due entirely to reading the publications of the work of Dr. A. S. Hoseason[1] in this country, and of Dr. Abraham Raab[2] in America that calcium was used.

I am indebted to Mr. James Kemble for permission to publish this case.

References.

1. *Brit. Journ. Anæsth.*, 1936, xiii, No. 4, 142.
2. *Current Researches in Anæsth. and Analg.*, 1936, xv, No. 6, 295.

A CASE ILLUSTRATING SOME OF THE WAYS IN WHICH CYCLOPROPANE DIFFERS FROM OTHER ANÆSTHETICS

By STANLEY ROWBOTHAM, M.D.,

Anæsthetist, King's College Hospital, etc.

THIS case is typical of many, but not by any means of all cases; it shows some of the ways in which cyclopropane differs from other anæsthetics.

The patient was a man of sparse build, aged 48 years, weighing 9 stone 2 lb. Operation: Partial gastrectomy for pyloric ulcer. Premedication: Omnopon gr. 9/30, 1½ hours before operation. (This represents gr. 1/30 per stone weight, a dosage which appears to give most constant results, and the interval of 1½ hours allows for its depressive action upon the respiratory centre to have passed off to a large degree). The blood-pressure before operation was 135/100.

Induction was started by filling the bag with oxygen and running in cyclopropane at the rate of 250 c.c. per minute. After two minutes this was increased for a short time to 500 c.c. per minute. Induction was quiet and uneventful, and light third-stage anæsthesia was obtained in 4½ minutes, there being no increased respiratory movement and no noticeable second stage. The patient was intubated and the mask held with harness so as to obtain a completely air-tight fit. The apparatus was set to deliver cyclopropane at 400 c.c. per minute and oxygen at 300 c.c. per minute.

The respiration now gradually became more depressed, until at 10 minutes it had finally ceased. (It is to be noted that the respiratory movements, although small, were not of the type associated with deep anæsthesia: that is to say, the whole chest as well as the epigastrium moved with each

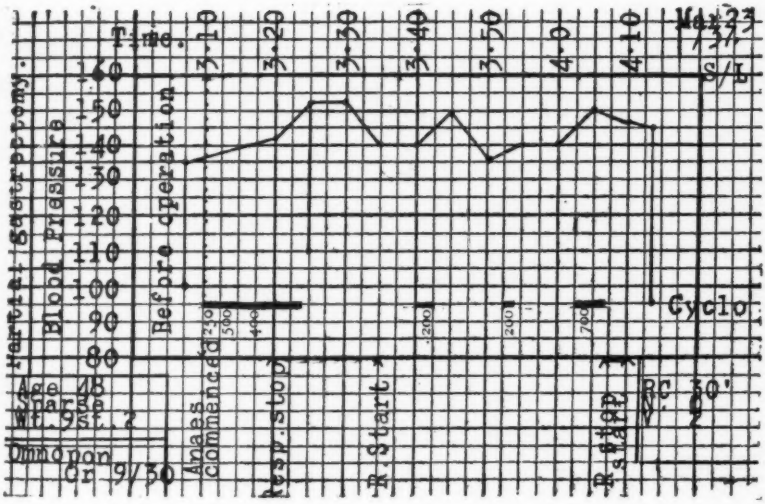
breath). At this point, although the patient was partially relaxed and his limbs were flaccid, nothing like the degree of relaxation required for abdominal section had been reached. To obtain this artificial respiration by rhythmically squeezing the bag about once every two seconds was resorted to, and good abdominal relaxation ensued in about three minutes; the patient's pulse and colour meanwhile remaining good. There was during this period no effort on his part at respiratory movement of any kind whatever. The concentration of cyclopropane in the bag was now weakened by running in oxygen, respiration being controlled by squeezing the bag. After a period of 12 minutes the patient commenced spontaneous respiratory movements and regular breathing was quickly established. The abdomen remained relaxed and small amounts of cyclopropane were run in from time to time to replace that which had been lost by diffusion, etc.

To close the abdomen it was desired to obtain the utmost relaxation possible, and more cyclopropane was therefore run into the bag. Again the respiration became shallow and finally ceased, but the anæsthetic was pushed by squeezing the bag. At the end it was quickly re-established by similar breathing control with a mixture of CO_2 and oxygen.

There are two other points to which I would like to draw attention. The systolic blood-pressure which was originally 135 rose to 152 soon after induction, thereafter it rose or fell roughly in proportion to the amount of cyclopropane which the patient was receiving. Without CO_2 absorption the systolic pressure tends to go higher, but in this case when the soda-lime canister was cut out in order to help stimulate respiration there was little alteration in the blood-pressure. Another point upon which most surgeons are agreed is that there is more bleeding with cyclopropane than with other anæsthetics; this may be due to the raised blood-pressure, but there have been cases in which the blood-pressure was not raised, but in which hæmorrhage was nevertheless profuse. Some observations are now being made on the blood and we may be able to give more information on his subject later.

The patient regained consciousness in half an hour, suffered no nausea, but vomited twice the same night.

The chief features of the case are represented on the chart.



THE DIPLOMA IN ANÆSTHETICS

THE following have been granted Diplomas in Anæsthetics by the Royal College of Physicians and Surgeons, after the May examination :—

C. D. Banes, R. P. Booth, Elva M. Chivers, R. T. V. Clarke, R. W. Cope, Herbert Curtis, G. D. Drury, A. C. Fraser, A. H. Galley, J. L. Hopkins, Ernest Landau, I. N. Lewis, H. J. V. Morton, A. H. Musgrove, J. A. V. Nicoll, W. Niven, Angela M. S. A. Ofenheim, G. S. W. Organe, F. H. Pratt, A. C. R. Rankin, O. N. Ransford, J. R. Ritchie, J. M. Savege (Major R.A.M.C.), H. R. Sheppard (Major R.A.M.C.), J. T. Turner, R. C. Walch, and Doris E. C. Walker.

At a meeting of the Council of the Royal College of Surgeons on June 10th Mr. H. E. G. Boyle, F.R.C.S., was appointed an examiner for the Diploma in the ensuing year.

ABSTRACTS.

On the anæsthesia of bromide of ethyl. A. JULIA in *Anesthés. et Analges.*, April 1933, p. 184.

The author believes that the objections, which resulted in the disuse of bromide of ethyl when it was tried as an anæsthetic up to about 1914, were chiefly due to impurities, not to the drug itself. He has now, through the work of Dumesuil, succeeded in getting pure bromide of ethyl. Dumesuil found as impurities alcohol, ether and bromoform. With the pure drug Julia is highly pleased. He finds quick anæsthesia and quick recovery. He finds the agent well suited both to those who are ill and those who are in good health. The only drawback is increased salivation. So far the author's experience has not been wide; he gives details of only 20 patients. The method of administration has been by simple open or closed apparatus.

Cyclopropane. H. G. GRIFFITH in *Canad. Med. Assoc. Journ.*, May 1937, p. 496.

The author gives an excellent summary of the history and present knowledge of cyclopropane including its chemistry and physiology. His clinical experience is based on over 2,500 personal administrations, and his patients ranged from an infant of seven days to a person of ninety years. In over 500 instances it was used endotracheally. He has observed no dangerous effects during the administrations. Dr. Griffith prefers to use small doses of avertin as a precursor to cyclopropane, but sometimes relies on nembutal with or without morphia. The average amount of the gas which he required for one hour of surgical anæsthesia was about one and a half gallons. The carbon-dioxide absorption technique was, of course, employed. Dr. Griffith does not find the contention upheld that cyclopropane increases bleeding. Nor does he think risks of explosion are formidable when a closed system is in use. "It would be just as difficult to cause an explosion by the use of an electric cautery in the abdomen as it would be to light the

gas in one's kitchen stove by striking a match at the other side of the room".

Anæsthesia by intravenous injection of 1-methyl-5, 5-allyl-150 prophyl barbituric acid. M. THALHEIMER in *Anæs. and Analges.*, April 1937, xvi, No. 2, p. 61.

Of the making of barbiturates, as of books, there is no end. The latest, a sodium salt of methyl allyl propyl barbituric acid prepared under the name of "Narconumal", has proved most satisfactory on some 500 occasions on which the author has used it. He prefers it to evipan, saying that it much less often causes excitement. He usually precedes the injection by one of morphia 20 minutes in advance. The narconumal is injected slowly, 2 c.c. per minute representing the maximum speed, the dose necessary may vary from 4 to 30 c.c. The dose is determined strictly by result produced, not by the operation to be performed. As a general rule 20 c.c. is regarded as the maximum dose. When supplementary anæsthetics are required the transition takes place quietly.

The prevention of collapse in spinal anæsthesia. W. DIETER in *Schmerz Narkose Anæsth.*, April 1937, p. 1.

The author believes that the blood-pressure fall during spinal analgesia is mostly due to paralysis of peripheral blood-vessels, but may also arise from affection of the vasomotor centre. Consequently its certain prevention requires a remedy which will control both these possibilities. Such a drug exists, he states, in cardiazol-ephedrin. For two days before operation 1 c.c. is injected intramuscularly night and morning. Another 1 c.c. is injected at the time of the spinal injection. After operation the injection is repeated every two hours during the next day. The author quotes cases in support of his contention and believes that the use of cardiazol-ephedrin, aided when necessary by cebion solution, makes spinal analgesia safe in gynæcology.

A new machine for gas and oxygen analgesia. A. BARR and H. TINDAL in *Lancet*, May 29th, p. 1,271.

The authors describe a machine for self-administration in labour. It consists of a rubber bag of ten gallons

capacity. Two tubes enter this, one leading direct from a nitrous oxide, the other from an oxygen cylinder. In order to measure the oxygen a one-gallon rubber bag which has a tap at each end, is connected up in the course of the tube coming from the oxygen cylinder. Two corrugated rubber links lead from the bag to the face-piece and directional valves are fitted so that the gas mixture is inspired through one tube and exhaled through the other. A valve is incorporated in the face-piece in such a way that when the plunger, which is spring-loaded, is depressed gas is permitted to enter the face-piece from the bag, and when it is released the flow of gas and oxygen is automatically stopped, air only being breathed. Before using the machine the bags are squeezed flat and then one gallon of oxygen let into the small bag. This is then emptied into the larger bag which is then filled to capacity with nitrous oxide. This larger bag now contains approximately 10 gallons of gas, 90 per cent N_2O and 10 per cent oxygen. The apparatus being ready for use whenever a pain approaches the patient expires to her fullest extent, places the face-piece over her nose and mouth, and, pressing the plunger valve, takes a long inspiration from the reservoir. She continues in this manner till the pain is over, when she releases the plunger and removes the face-piece. One filling of the reservoir lasts from half to two and a half hours, depending on the frequency of the pains. Somebody is needed to refill the bag from time to time. One hundred patients were treated with the apparatus. Eight of these were unsatisfactory through lack of self-control or inability to obey instructions for using the machine. None of these were in private practice. Twenty-nine patients were unaware of the birth of the child and 48 had very good relief from pain although they felt the natural birth. There was no untoward cyanosis and the child was never affected.

Anæsthesia for intra-cranial operation. P. AYRE in *Lancet*, March 6th, 1937, p. 561.

Believing that vascular oozing is the surgeon's chief obstacle in neurological operations, the author has devised a simple modification in his anæsthetic technique which is,

he maintains, successful in reducing bleeding during operation. Only light premedication (omnopon 1/3, scopolamine 1/150) is employed. A Magill wide-bore endotracheal tube is passed. To this the special bit of apparatus is attached. It consists simply of a metal T-piece connected by a short piece of rubber tubing and a metal angle-piece. One limb of the T-piece is connected to some apparatus delivering a continuous flow of oxygen with ether or chloroform vapour. The other limb is open to the outside air. A length of tubing not less than one centimetre in inside diameter is attached to the outside end and should be stuck with adhesive plaster to some convenient part of the anæsthetic table. A thin piece of gauze attached near the outside end of the tube, being blown to and fro by the patient's respirations, acts as a useful indicator. By this method the patient breathes fresh air, supplemented by a continuous flow of oxygen at the rate of three or four litres a minute to which is added the minimal amount of ether or chloroform vapour to maintain anæsthesia. The conditions of respiration are physiological, so there is both quiet breathing and absence of vascular congestion.

Effect of anæsthetics on lymphatic absorption from peritoneal cavity. H. A. MENGLE in *Archives of Surgery*, May 1937, p. 839.

The author points out that a vital factor in peritonitis is the accompanying toxæmia. Therefore anything which promotes or inhibits absorption of toxic matter from within the abdomen must aggravate or reduce the danger of the condition. He instituted experiments to find out if possible what part, if any, the anæsthetic plays in delaying or in hastening absorption of toxic matter in patients suffering from peritonitis. It was soon found that lymphatic absorption was chiefly by the diaphragmatic route. The venules of the omentum appeared, however, to be the chief avenue of exit for toxic material in the peritoneal cavity. The activity of the diaphragmatic lymphatic absorption varied with the respiratory activity. It was greater when the breathing was increased. Consequently ether anæsthesia and any other form of anæsthesia which is accompanied

by exaggerated respiratory movements promotes absorption of toxic matter from the abdomen of a patient suffering from peritonitis. The author concludes that the method of choice for operations in the presence of peritonitis is local or regional block or low spinal anæsthesia.

Encephalography with anæsthetic gases. R. B. AIRD in *Archives of Surgery*, May 1937, p. 853.

There appear to be certain advantages in using anæsthetic gases rather than air or oxygen for encephalography. Nitrous oxide and ethylene both proved safe agents to employ and gave good roentographic results. There is, however, the drawback to nitrous oxide that its great solubility shortens the time too much during which good views could be obtained. Ethylene provided 40 or 50 minutes during which this was possible. Moreover, the slight sedative effect of the ethylene lessened the severity of the immediate reaction after encephalography. A considerable shortening resulted of the stay necessary in hospital. In children only induction of anæsthesia was needed, the sedative effect of the ethylene carrying the patient quietly through the procedure. Pure ethylene gas was used and this is not explosive. For adults avertin injection was employed prior to the encephalography. Several patients were relieved of their symptoms after this procedure.

CORRESPONDENCE.

NASAL ENDO-TRACHEAL ANÆSTHESIA.

To the Editor, British Journal of Anæsthesia.

Dear Sir,—I would be grateful if you would allow me to reply to some of the points raised by your correspondents on the question of pulmonary complications following nasal endotracheal anæsthesia.

With regard to the two fatal cases described, as far as I have been able to ascertain, not having given the anæthetics myself, cocaine was not used to spray the nose before induction of anæsthesia. With regard to the suggestion of possible error in technique, as these figures included the work at several different hospitals, it is hardly likely that the same errors in technique would occur in each hospital. On analysis of my own cases I have found that there is a definite relation between the pulmonary morbidity and the number of attempts made to pass the tube. The incidence of pulmonary complications when the tube entered the trachea at the first attempt was 3.9 per cent; where two or more attempts were made before passage of the tube the incidence of complications rose to 10.7 per cent. It would be interesting to know whether this increase has been noted by other anæsthetists. In conclusion, I should like to point out that I did not endeavour to belittle the nasal endotracheal method in my article, but rather attempted to show the possible dangers of its indiscriminate use.

I am,

Yours faithfully,

C. J. Massey Dawkins.

Obituary.

Harry P. Fairlie.

AFTER a long and painful illness Harry Prescot Fairlie, M.D., Anæsthetist Western Infirmary, Glasgow, etc., died in March, and with his passing went one of the finest personalities that Glasgow has given to the medical profession. It is difficult to talk or write about Fairlie without wallowing in superlatives.

A slender slip of a man, with rather a sad expression as a habit, his life was devoted to the practice of anæsthetics, and, that he was the ideal anæsthetist no one who ever had the privilege of working with him ever doubted.

In addition to an uncanny efficiency in his technique, he was the possessor, in a very high degree, of that God-given gift of giving, merely by his presence, that calm confidence to patient, surgeon, and theatre staff, which makes all the difference in an operating theatre.

I am sure I can say, without fear of contradiction, that no medical man in the history of Glasgow was ever more beloved and respected by profession and public alike than Harry Fairlie, and he thoroughly deserved all the affection that was his. I had the happiness to be associated with him professionally almost every day during the last ten years, and a good many of our leisure hours were also spent together. It was a great privilege to have known and loved him.

I used to wonder what it was in his make-up that made him so universally popular, for he was no showman, and he was quiet almost to the verge of taciturnity.

I came to the conclusion that the keynotes of his character were modesty, simplicity, selflessness, and a great faith.

In his quiet way he was a very happy man, happy in his home life and in his work, and play. One could not

be in his presence for long without realising that he was made of finer clay than most, and this was reflected in all his ways. He had a good sense of humour and it took small provocation to bring a Puck-like smile to his usually grave features.

He loved the life of the countryside and had a passion for mountains and birds, whilst he was a very useful disciple of Izaak Walton. Music, the arts, and general reading held a lot of interest for him, and during the last few years he was a Rem bridge player.

To a great anæsthetist, a loyal friend, and a wonderful personality, I pay this tribute. We are rich in having known him, and the world is infinitely poorer for his early going.

G. B. BRAND.